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VAN DIE REDAKSIE

SKOK BY AKUTE HARTVERSTOPPING

Die siektebeheer van akute verstopping van die hart is grotendeels toegespits op die voorkoming en behandeling van die komplikasies. Omtrent die helfte van die pasiënte wat die ramp self oorleef, sal rustig herstel. By die ander word die herstel bemoeilik deur talle komplikasies—hartversaking, aritmie, propvorming, hartbreuk en skok. Die gebruiklike behandeling vir hartversaking is met digitalis, soutonthouding en diuretika. Terloops kan dit weer beklemtoon word dat matige dosisse digitalis *wel* by hartverstopping gebruik kan word. Die aritmies word volgens hulle oorsprong behandel; die meeste boesem-aritmies word doeltreffend met digitalis behandel, terwyl dié wat in die kamers ontstaan met chinidien of procaine amied beheer word. Procaine amied mag slegs in noodgevalle binneaars gegee word, en dan moet dit met groot versigtigheid geskied omdat dit die bloeddruk verlaag.

Daar is geen bepaalde behandeling vir die komplikasies van propvorming en hartbreuk nie, maar hulle voorkoms kan verminder word met geskikte voorbehoedende behandeling. Middels teen bloedstolling is aangewese vir die voorkoming van klontvorming. Hoewel die doeltreffendheid van hierdie behandeling grondig bevestig is, kan dit nogtans op sigself verantwoordelik wees vir 1·7 persent van die sterftes weens bloeding.[1] Baie gesaghebbendes behandel dus nie hulle ligte gevalle, by wie propvorming seldsaam is, met koaguleringverhoedmiddels nie. Hulle beperk die gebruik daarvan tot die ‚kwaai gevalle'[2] wat gekenmerk word deur 'n geskiedenis van vorige verstopping, of wat gepaard gaan met onbeheerbare pyn, hartversaking of skok, waar propvorming betreklik dikwels voorkom.

Die gevaar van hartbreuk kan verminder word as die genesing van die wond in die hartspier vergemaklik word. 'n Streng ruskuur moet toegepas word gedurende die eerste 2 weke, wanneer nekrose van die spiere en akute ontstekingsinsyfering in die selle die weefselbeeld oorheers, en wanneer hartbreuk die meeste voorkom. Teen die einde van die tweede week, wanneer die bindweefsel-vesels al goed herstel, word die pasiënt toegelaat om homself te voed en self sy ligte toilet te ver-

EDITORIAL

SHOCK IN ACUTE CARDIAC INFARCTION

The management of acute cardiac infarction is largely concerned with the prevention and treatment of its complications. Of the patients who survive the immediate catastrophe, about half will recover uneventfully. In the others, the road to recovery is beset with complications—heart failure, arrhythmias, thrombo-embolism, cardiac rupture and shock. Cardiac failure is treated conventionally with digitalis, salt restriction and diuretics. In passing it is well to emphasize again that digitalis in moderate dose is *not* contra-indicated in cardiac infarction. The treatment of the arrhythmias will vary with their site of origin: most supraventricular arrhythmias are effectively treated with digitalis; those arising in the ventricles are controlled by quinidine or procaine amide. Intravenous procaine amide should be given only in emergency and with due caution because of its hypotensive action.

Thrombo-embolic complications and cardiac rupture have no specific treatment, but their incidence may be reduced by the appropriate prophylactic management. Anticoagulants are indicated for the prevention of thrombo-embolism. Although the efficacy of this treatment has been clearly substantiated, it may itself be responsible for a 1·7% mortality from haemorrhage.[1] Many authorities, therefore, do not administer anticoagulants to mild cases, in which thrombo-embolism is rare, but restrict their use to those 'bad cases',[2] characterized by a history of previous infarction, or accompanied by intractable pain, cardiac failure or shock, in which thrombo-embolism is relatively common.

The danger of cardiac rupture may be lessened by facilitating adequate healing of the myocardial wound. In the first 2 weeks, when muscle necrosis and acute inflammatory-cell infiltration dominate the histological scene and cardiac rupture is most common,[3] strict rest is enforced. At the end of the second week when

sorg. Na 4 weke, wanneer die meeste pasiënte mag opstaan, is baie lymstof reeds neergelê, maar die pasiënte moet hulle ten minste nog 2 maande lank baie stil hou terwyl die littekenweefsel stewig aangroei.[4]

‚Hartskok' ná hartverstopping is 'n toestand waarby die sistoliese bloeddruk gedurig laer as 80 mm. Hg is, hoewel daar nie ander bloeddrukverlagende omstandighede soos vinnige hartslagonreëlmatigheid, longverstopping, diabetiese suurvergiftiging of 'n beskadiging van die harsingbloedvate ens.[5] is nie. Dit moet onderskei word van die kortstondige bloeddrukverlaging wat tydens die eintlike verstopping plaasvind en wat verbygaan sodra die pyn deur morfien of 'n soortegelyke middel verlig word. Die patogenese van hartskok word nog nie duidelik verstaan nie. Dit word hoofsaaklik veroorsaak deur die onvermoë van die sentrale pompwerking van die beskadigde hartspier om genoeg bloed uit die hart te stoot. Dit word dikwels vererger deur die afwesigheid van 'n vergoedende vermeerdering in die weerstand van die periferale bloedvate, d.w.s. deur 'n instorting van die periferale bloedsomloop. Dit kan ook verder vererger word deur oormatige toediening van morfien, of as die pasiënt in 'n regop houding met die bene laag verpleeg word. Hoe laer die bloeddruk, hoe swakker is die bloedsomloop in die kransslagaar en hoe groter is die verspreiding van die verstopping; 'n noodlottige kringloop word dus ingestel wat die hart se kragvermoë meer en meer benadeel. Vroegtydige en kragdadige behandeling is onontbeerlik as die gewone 80-90 persent kans op die dood vermy wil word.

Behandeling is eerstens gemik op die beperking van die omvang van die hartspier se beskadiging. Hierdie doel word bereik deur die oordeelkundige gebruik van suurstof om die plaaslike bloedloosheid van die hartspier te verbeter; deur die vroegtydige gebruik van binneaarse heparien om die propvormingproses te stuit, en deur die spoedige herstelling van 'n bevredigende diastoliese bloeddruk. Tweedens word daar deur die binneaarse toediening van herhaalde klein dosisse van 'n vinnig-werkende digitalis-glikosied (bv. Ouabain),[6] probeer om die hart se uitstootvermoë te vermeerder. Op hierdie manier word die saamtrekbaarheid van die hartspier vermeerder, en as die digitalis in klein dosisse toegedien word, is daar min kans dat die hartspier te prikkelbaar sal word. Binneaarse[7] of binneslagaarse[8] oortappings van volle bloed om die hart se werkvolume te vermeerder het ook hul voorstanders, en 'n paar dramatiese verbeterings is reeds te boek gestel. Maar by hartversaking met bloedstuwing, wat dikwels 'n verdere komplikasie by hartskok is, kan die oortappings nie in groot genoeg hoeveelhede of teen 'n bevredigende snelheid gegee word nie, en op sigself het hulle nie juis veel gedoen om die sterftesyfer te verminder nie.

Ten laaste kan die behandeling daarop gemik wees om die periferale weerstand te vermeerder, en dit is blykbaar die doeltreffendste van die beskikbare prosedures. Noradrenalien of een van die verwante sintetiese amien-simpatikomimetika word vir hierdie doel gebruik. Hierdie stowwe het hoofsaaklik 'n periferale bloeddrukverhogende werking, maar Sarnoff en sy medewerkers[9] het onlangs aan die hand gegee dat

fibroblastic repair is well under way, the patient is permitted to feed himself and to attend to minor toilet procedures. Much collagen has already been laid after 4 weeks, when most cases are allowed up, but restriction of activity for at least 2 more months is required while sound scarring is established.[4]

'Cardiogenic shock' after cardiac infarction is a state in which the systolic blood pressure is persistently below 80 mm. Hg in the absence of other hypotensive circumstances such as rapid cardiac arrhythmia, pulmonary infarction, diabetic acidosis, cerebral vascular accident, etc.[5] It is to be distinguished from the transient fall in blood pressure which occurs at the time of infarction and which passes when morphine or a similar drug relieves the pain. The pathogenesis of cardiogenic shock is not clearly understood. It is mainly due to failure of the central pumping action of the damaged myocardium to maintain an adequate cardiac output. It is often aggravated by the absence of a compensatory increase in peripheral vascular resistance; that is, by peripheral circulatory collapse. It may be further aggravated by the administration of too much morphine, or by nursing the patient in an upright position with the legs down. The lower the blood pressure, the poorer is the coronary circulation and the greater the spread of the infarction; thus a vicious circle is established to the progressive detriment of the cardiac output. Early and vigorous treatment is essential if the usual 80-90% chance of death is to be avoided.

Treatment first aims at limiting the extent of the myocardial damage. This is achieved by the judicious use of oxygen to reduce the myocardial ischaemia; by the early use of intravenous heparin to halt the thrombolic process and by the early restoration of an adequate diastolic blood-pressure. Secondly an attempt is made to increase the cardiac output by giving repeated small doses of a quick-acting digitalis glycoside (e.g. Ouabain) intravenously.[6] In this way the contractility of the myocardium is increased and if the digitalis is given in small doses the danger of increasing myocardial irritability is minimal. Intravenous[7] or intra-arterial[8] transfusions of whole blood to increase cardiac output have their advocates and some dramatic responses to these procedures are on record. But transfusions cannot be given in effective volume or at adequate rate in the presence of congestive heart failure, which often complicates cardiogenic shock, and on their own they have done little to lower the mortality rate.

Finally, treatment may aim at elevating the peripheral resistance, and this seems to be the most effective of the available procedures. Nor-adrenaline, or a related synthetic sympathicomimetic amine is used for the purpose. These substances have a predominantly peripheral vasopressor action, but Sarnoff and his

hulle in 'n mindere mate ook direk die saamtrekbaarheid van die hartspier kan stimuleer. Hierdie middels vermeerder nie die hartspier se prikkelbaarheid nie, en die hart se bloedvolume word vermeerder sonder dat die kransslagaar meer bloed per eenheid arbeid moet lewer. Noradrenalien self is die kragtigste van hierdie bloeddrukverhogende middels; dit word as 'n stadige binneaarse inspuiting van 5 persent dekstrose in water bevattende 'n 4 mg.-noradrenalienbasis gegee. Dit is noodsaaklik dat die bloeddruk noukeurig dopgehou word gedurende die inspuiting, want as dit te veel of te vinnig verhoog word, kan longedeem intree. Dit is 'n vereiste dat hierdie prosedure met die grootste noulettendheid uitgevoer word, want selfs 'n geringe lek uit die bloedvat kan ernstige weefselversterwing veroorsaak.[10]

Weens hierdie gevare is die sintetiese drukverhogende amiene ingevoer; hulle werking is baie minder drasties, en hulle kan in herhaaldelike spier- en aarinspuitings toegedien word. Hulle sluit die volgende middels in: fenielefrien waterstofchloried (‚neo-synephrine'), hidroksiamfetamien (‚paredrine'), mefentermien (‚wyamine') en isopropielnoradrenalien (‚isuprel'). Laasgenoemde is veral waardevol by gevalle waar hartblokkade ook voorkom.[5] Mits die skoktoestand kort tevore eers ingetree het, is dit in die praktyk gerieflik om eers die werking van die sintetiese drukverhogende middels uit te toets. As daar egter nie 'n dadelike en blywende reaksie is nie, moet 'n noradrenaliendrup dadelik ingestel word. Die spoed van toediening sal bepaal word deur die gevolglike drukverhoging, maar oor die algemeen kan dit aangeneem word dat die uiteindelike kans op oorlewing gering is[5] as méér as 1 mg. noradrenalien per uur nodig is om 'n uitwerking te hê. Nieteenstaande sy beperkings en gevare, is hierdie soort terapie ongetwyfeld waardevol, en die gebruik daarvan het oor die algemeen die hartskok-sterftesyfer met 20 persent verminder.[11]

1. Wright, I. S., Beck, D. F. en Marple, C. D. (1954): Lancet, **1,** 92.
2. Russek, H. L. en Zoman, B. L. (1952): Amer. J. Med. Sci., **224,** 496.
3. Wessler, S., Zoll, P. M. en Schlesinger, M. J. (1952): Circulation, **6,** 334.
4. Mallory, G. K., White, P. D. en Salcedo-Salager, J. (1939): Amer. Heart J., **18,** 647.
5. Binder, M. J., Ryan, J. A., Marcus, S., Mugler, F., Strange, D. en Agress, C. M. (1955): Amer. J. Med., **18,** 622.
6. Gorlin, R. en Robin, E. D. (1955): Brit. Med. J., **1,** 937.
7. Sampson, J. J. en Singer, I. M. (1949): Amer. Heart J., **38,** 54.
8. Silver, E. N., Levin, D. B., Becker, G. H. en Levy, R. C. (1951): J. Amer. Med. Assoc., **147,** 1626.
9. Sarnoff, S. J., Case, R. B., Berglund, E. en Sarnoff, L. C. (1954): Circulation, **10,** 84.
10. Bifulco, E. (1953): J. Amer. Med. Assoc., **152,** 1554.
11. Andrus, J. C. (1955): Minnesota Med., **38,** 888.

associates[9] have recently suggested that to a lesser extent they may also stimulate myocardial contractility directly. These drugs do not increase myocardial irritability and the cardiac output is enhanced without demanding increased coronary flow per unit work. Noradrenaline itself is the most potent of these pressor drugs; it is given by slow intravenous infusion of 5% dextrose in water, containing 4 mg. of nor-adrenaline base. Careful monitoring of the blood pressure is essential during the infusion because, if it is elevated too far or too fast, pulmonary oedema may supervene. A scrupulous technique is also necessary because even slight extravascular leak may cause intense tissue necrosis.[10]

Because of these hazards the synthetic pressor amines have been introduced; they are far less drastic in their effects and may be given by repeated intramuscular or intravenous injection. They include phenylephrine hydrochloride ('neo-synephrine'), hydroxyamphetamine ('paredrine'), mephentermine ('wyamine'), and isopropylnoradrenaline ('isuprel'). The latter is of particular value in cases associated with heart block.[5] In practice, provided the shock is of recent onset, it is convenient to try the effect of the synthetic pressor drugs first. Unless there is a prompt and sustained response, however, a nor-adrenaline drip should be instituted immediately. The speed of infusion will depend on its pressor effect, but in general, if more than 1 mg. of nor-adrenaline per hour is required to obtain an effect, the ultimate chances of survival are slight.[5] Despite its limitations and dangers, this form of therapy is undoubtedly valuable and an over-all reduction of 20% in the mortality from cardiogenic shock has been achieved by its use.[11]

1. Wright, I. S., Beck, D. F., and Marple, C. D. (1954): Lancet, **1,** 92.
2. Russek, H. L., and Zohman, B. L. (1952): Amer. J. Med. Sci., **224,** 496.
3. Wessler, S., Zoll, P. M., and Schlesinger, M. J. (1952): Circulation, **6,** 334.
4. Mallory, G. K., White, P. D., and Salcedo-Salager, J. (1939): Amer. Heart J., **18,** 647.
5. Binder, M. J., Ryan, J. A., Marcus, S., Mugler, F., Strange, D., and Agress, C. M. (1955): Amer. J. Med., **18,** 622.
6. Gorlin, R. and Robin, E. D. (1955): Brit. Med. J., **1,** 937.
7. Sampson, J. J. and Singer, I. M. (1949): Amer. Heart J., **38,** 54.
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9. Sarnoff, S. J., Case, R. B., Berglund, E., and Sarnoff, L. C. (1954): Circulation, **10,** 84.
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UNION DEPARTMENT OF HEALTH BULLETIN

Union Department of Health Bulletin. Report for the 7 days ended 3 April 1956.

Plague. Smallpox. Typhus Fever. Nil.

Epidemic Diseases in other countries

Plague. Nil.

Cholera in Calcutta (India); Chittagong, Dacca (Pakistan).

Smallpox in Kandahar (Afghanistan); Moulmein, Rangoon (Burma); Phnom-Penh (Cambodia); Ahmedabad, Bombay, Calcutta, Delhi, Madras, Pondicherry, Visakhapatnam (India); Chittagong, Dacca, Lahore (Pakistan); Saigon-Cholon, Tourane (Danang) (Viêt-Nam); Nairobi (Kenya).

Typhus Fever in Kabul (Afghanistan); Alexandria (Egypt).

SOME OBSERVATIONS ON THE DEVELOPMENT OF KWASHIORKOR

A STUDY OF 205 CASES

P. J. PRETORIUS, M.B., CH.B. (PRETORIA)*
and
J. G. A. DAVEL, B.M. (OXFD), F.R.C.S.E., M.R.C.P.E., D.C.H.
Department of Paediatrics, University of Pretoria, Pretoria
and
J. N. COETZEE, M.D. (CAPE TOWN)
Department of Bacteriology, University of Pretoria, Pretoria

Kwashiorkor is at present considered to result from protein deficiency[6] in the diet of the infant and toddler, and it is known that the substances responsible for its cure are to be found in skimmed cow's milk.[3] The purpose of this study is to determine the importance of various possible etiological factors.

The 205 cases of kwashiorkor were admitted to one of the Bantu children's wards of the Pretoria Hospital during the period January 1954 to December 1955. The schedule of treatment has been outlined in previous communications.[3, 14] There was a 15% mortality (31 deaths) but, if fatalities occurring within 48 hours are omitted, the mortality rate is reduced to 9%.

I. *Age and Sex*

The age incidence is indicated in Table I, and, although the parents are often undecided about the age of the infant, the figures presented, correspond well with those of other authors;[17] 67% of cases were between 1

TABLE I. AGE DISTRIBUTION OF 205 CASES

Age (years)	*No. of Cases*
0—1	26 (12·7%)
1—2	138 (67·3%)
2—3	30 (14·6%)
Over 3	11 (5·4%)

and 2 years and 82% between 1 and 3. The age limits for admission were 6 months and 5 years respectively. The opinion has been expressed[23] that this syndrome occurs more frequently in boys than in girls and that girls are usually older when the syndrome develops. Of our cases 110 (53·7%) were boys, and the average age at admission was 22 months for boys and 21 months for girls, The series is too small to admit of a definite conclusion.

II. *Race*

In this series 200 were Bantu children and 5 were Coloured. The Coloured population of Pretoria is small. There were no Indian patients in spite of the relatively large Indian population. European cases are rare; usually less than 4 are seen per year.

III. *Diet*

1. *Carbohydrate.* The main article of diet of the Bantu in this region is mealie-meal porridge and the children received as much of it as they desired 2 or 3 times a day. In addition some received a slice of white bread 2 or 3 times a week.

2. *Protein:*

(*a*) Meat. Few families could afford meat more than once a week, with the result that the children received a minimal amount of this commodity.

(*b*) Milk. The majority of children received no milk. The few lucky ones got a maximum of 2 or 3 cups per week.

(*c*) Fish. The Bantu in Pretoria rarely if ever eat fish.

(*d*) Eggs. The children seldom get eggs to eat. Some families have a few chickens but the men often consume the eggs that are not sold. Some of the Bantu tribes apparently believe that eggs are bad for children and women.

3. *Vegetables and Fruit.* Most families have vegetables only with their Sunday dinner. The children seldom get fruit, but oranges are occasionally available.

4. *Breast-feeding.* 12 cases were still being partially breast-fed on admission. One baby of 15 months was entirely breast-fed but, as a test feed was not done, it is difficult to substantiate our impression that the breast was merely proffered as a dummy.

5. *Weaning.* The disease usually developed 1-6 months after cessation of breast-feeding. This corresponds with Brock and Autret's observation[2] that the syndrome usually develops during the late breast-fed, weaning and post-weaning periods.

IV. *The Social Background*

The incidence of kwashiorkor parallels that of poverty, with ignorance adding its quota.

1. *The Income of the Head of the Family.* In only 87 cases was the mother aware of her husband's income. The average was £10 per month with a scatter of £2 to £36. Approximately 1/3rd of the mothers also worked Many of these had been deserted and were employed as domestic servants earning £2-£5 per month with board and lodging for themselves only.

2. *The Size of the Family.* The average number of living children was 2·2 per family. Many families had lost one or more children. Furthermore, although the number of living children was so surprisingly small many fathers also supported other relatives.

3. *The Number of Fathers actually Supporting their*

* Present address: National Nutrition Research Institute, Council for Scientific and Industrial Research, Pretoria.

Families. In only 120 cases information regarding the fathers' activities could be obtained: 25 had disappeared, 7 had died, 3 were unemployed and 1 was in a mental hospital, while 5 mothers were unmarried, Thus, in only 79 families were the fathers responsible for their support and in the remaining cases the mothers were forced to leave the children in the care of relatives or friends while they went out to earn money.

4. *Number of Cases where Relatives were the sole Support.* In 43 out of the 200 Bantu cases, relatives were totally responsible for the children. Usually it was one or other of the grandparent pair. This occurred especially where both parents were working or where the father had deserted. It can readily be conceived that the grandparents either through poverty, ignorance, superstition or old age failed to give the children the necessary care. Trowell, Davies and Dean[18] mentioned the possibility of the separation of child and mother being etiologically important, in that the loss of security so produced could cause anorexia. It is accepted[7] that the absence of the Mother-figure not only leads to psychic trauma but also to physical retardation. Whether this is important in the etiology of kwashiorkor remains an open question.

5. *Housing.* The housing is extremely poor. Often the family as well as relatives or even friends share 1 or 2 rooms. Sachs[15] emphasized the importance of environment: he points out that with a poor diet a mild degree of malnutrition may result, but if the environment is poor, especially under unhygienic conditions, gastro-enteritis followed by nutritional oedema may occur.

V. *The Role of Infection*

On admission, many of the cases show evidence of infection. This may play a part in the development of the syndrome, or it may be a precipitating factor. Another possibility is that it develops as a result of lowered resistance.

1. *Infection of the Respiratory System:*

(*a*) Ear, Nose and Throat. A large proportion of the cases had a nasal discharge, which cleared with recovery. At times the throat was red and injected. The appearance of the tympanic membrane was characteristic. In practically every instance it was dull-grey in appearance, with no light reflex. Malnutrition may be the cause, but we usually regard it as evidence of low-grade infection. Of the 13 cases in which post-mortem examination was done, 3 showed mastoiditis and 2 bilateral otitis media. We feel that middle-ear infection in cases of kwashiorkor should receive more attention.

(*b*) Pulmonary Infection (non-tuberculous). On clinical examination 16 cases were diagnosed as having bronchitis or pneumonia. Roentgen examination revealed a further 11 cases of pneumonia. These were all Mantoux-negative.

(*c*) Tuberculosis. In 190 cases Roentgen photos of the lungs were taken and a Mantoux test (PPD second strength) done soon after admission. Only 7 cases were Mantoux-positive, and 4 of these had radiological evidence of tuberculosis. Roentgen examination revealed lesions suggestive of pulmonary tuberculosis in a further 20 cases. The commonest lesion was a primary complex, but collapse, broncho-phneumonia and cavity formation were also encountered. One case had tuberculous spondylitis. At post-mortem examination 1 further case of tuberculosis was discovered.

2. *Congenital Syphilis.* Kolmer complement fixation and Price precipitation tests were done on 200 of the cases; 192 were frankly negative while 3 cases were positive (qualitative precipitation-test positive and Kolmer test 128 units). The remaining 5 cases showed various discrepancies and were of such low titres that the serological diagnosis was in doubt. None of the cases showed any clinical signs of congenital syphilis. Janssen and le Roux[9] obtained positive results in 5 out of 101 cases. Gillman and Gillman[8] found a strongly positive Wassermann reaction in 16 out of 60 adult 'pellagrins' and in 2 out of 22 'infantile pellagrins'. Harris (quoted by Gillman and Gillman[8]) reported positive W.R. in many Negro 'pellagrins' in America.

3. *Gastro-intestinal Infections:*

(*a*) History of Diarrhoea. Out of 200 cases 95 gave a history of diarrhoea, but in many cases the history was not reliable. Unfortunately it was not possible to establish the presence of diarrhoea in all cases, but the majority of those examined had loose stools.

(*b*) Investigation of Faeces. The faeces of 180 cases of kwashiorkor were examined:

(i) Macroscopic appearance. In 72 cases the specimens were loose and watery and in many instances green.

(ii) Microscopic examination. This was done according to the methods of Bates and Alberto[1] and Joseph,[10] and 141 specimens showed no abnormality. The remaining 39 cases showed the presence of scanty pus cells and/or red blood-cells. *Trichomonas hominis* was present in abundance in 12 of the 39 cases. In one of these cases the vegetative form of *Balantidium coli* was found, in another that of *Giardia lamblia* and in a 3rd case encysted forms of *Entamoeba histolytica* were present. Ova of *Ascaris lumbricoides* were present in one case.

(iii) Pathogenic organisms. The cause of diarrhoea in kwashiorkor is as yet uncertain. Trowell, Davies and Dean[19] mention the possibility that certain types of diet may play a role, e.g. those that contain an excess of cane sugar or mealie-meal. It has however been demonstrated[16] that these patients have a diminished secretion of digestive enzymes, and that this may be an important cause of diarrhoea.[21] To establish whether infection plays a role the faeces of 180 cases were cultured for shigella and salmonella organisms according to the method outlined by Coetzee and Scott.[5] In 10 cases various shigellas (mostly of the flexner type) were isolated, while in 3 cases salmonellas were cultured. In 1954 4 cases were investigated for the presence of 'pathogenic' *Escherichia coli.* These were present in all 4 cases (3 had double infections); the types isolated were 055B5, 0119B?, 0128B12, and 026B6. Coetzee and Pretorius[4] demonstrated that these strains of *E. coli* are frequently found in cases of gastro-enteritis in Pretoria, and there is no

reason to believe that cases of kwashiorkor are less susceptible to infective diarrhoea. Mild cases of malnutrition may possibly develop serious symptoms when gastro-enteritis supervenes. It has been shown that the 'pathogenic' strains of *E. coli*, occur in healthy carriers[13] and the possibility that gastro-enteritis will develop if their resistance is lowered has been mentioned.[24] Babies harbouring these strains develop diarrhoea on a change of diet or on contracting an upper-respiratory-tract infection.[24] It appears reasonable to consider that the resistance of cases of kwashiorkor is lowered, especially as many of them suffer from upper-respiratory-tract infections, and we regard it as important to determine the role of *E. coli* in the occurrence of diarrhoea in cases of this disease. We are at present engaged on a study of this.

4. *Infection of the Urinary Tract:*

The urine of 185 cases was examined microscopically after centrifugation and for the presence of albumin. Albuminuria was found in 75 (40%) (see Table II)

TABLE II. ALBUMINURIA

Albumin	*No. of Cases*
Negative	110 (60%)
Trace	33
1+	13
2+	14
3+	9
4+	6
Total	185

and this agrees with the experience of others[9, 22] that albuminuria occurs frequently. Pus cells were seen in 124, usually 2-10 per high-power field, while an additional 16 had a frank pyuria. In 55 of the 124 with pus cells red blood-cells were also present (2-8 per high-power field). No bilharzia ova were ever detected. In 15 cases (8%) the urine specimens contained casts; 7 were hyaline, 5 granular and the specimens

TABLE III. RESULTS OF URINE CULTURES

Organisms	*No. of Isolations*
Sterile	10
Escherichia coli	29
Proteus vulgaris	5
Proteus mirabilis	2
Proteus rettgeri	1
Pseudomonas aeroginosa	6
Micrococcus pyogenes (Coagulase-positive) ..	5
Achromobacter	5
Paracolon	4
Alpha haemolytic streptococci	2
Total	69

of 3 cases contained both varieties. In 54 cases with microscopic evidence of urinary infection the urine was cultured. Results are set out in Table III. In some instances more than one organism was isolated. The well-known difficulty of obtaining an aseptic specimen from young children renders the interpretation of the findings difficult but we feel that urinary infection occurs fairly frequently in these patients, although the relationship to kwashiorkor is undetermined.

VI. *Parasitism*

Formerly, in certain parts of the world, kwashiorkor was thought to occur entirely as a result of parasite infestation,[20] and even today some authors[6, 11] consider it important etiologically. In some parts the incidence of parasitism is low and its importance in kwashiorkor is discounted.[12]

In Pretoria and environs malaria does not occur to any great extent and none of our cases showed evidence of this disease. The presence of helminths was established from the history in 16 cases and in one instance ova of *Ascaris lumbricoides* were seen in a microscopic examination. Parasitism does not appear to play an important role in this area.

SUMMARY AND DEDUCTIONS

Kwashiorkor is a deficiency syndrome, certainly of protein and possibly of other nutrients as well. Protein foods are expensive, so that where socio-economic conditions force the purchase of the cheaper carbohydrate foods (mealie-meal in this area) protein deficiency must arise.

The moment the supply of breast-milk diminishes or the baby is weaned, the stage is set for the development of kwashiorkor. These patients suffer great disabilities. Their digestive capacity is impared owing to a diminution of enzyme secretion—a possible cause of the diarrhoea. Secondly, they certainly seem very prone to infection. Whether infection plays an etiological role is undecided but it seems reasonable to consider that by creating greater demands for nutrients, especially proteins, it is important in the etiology of kwashiorkor.

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PERINATAL MORTALITY*

L. G. R. VAN DONGEN, M.Sc., M.D. (RAND), M.R.C.O.G.
University of the Witwatersrand

The stillbirth rate is often taken as an index of the standard of obstetric practice for any institution or country. Though this index is one of the best we have, many other factors than the purely obstetric are involved in the problem of stillbirths. It is known that the age of the mother influences the stillbirth rate considerably; the older the mother the higher the stillbirth rate. Parity also plays an important part, the stillbirth rate being higher with the first infant than with the second, and once the patient has become a grande multipara the stillbirth rate rises very markedly. Again it has been shown that a rapid succession of pregnancies is associated with a higher incidence of stillbirths than where the births are spaced at intervals of 2 years. Good and adequate nutrition of the mother is known to reduce the incidence of stillbirths and also of premature labours. The importance of nutrition was clearly demonstrated during the siege of Leningrad in 1942. Here the stillbirth rate doubled itself and the incidence of prematurity rose from 6·5% to 41·2%. General social factors also play some part, as revealed by a study of the British Registrar-General's report, which shows a much higher stillbirth rate among the families of unskilled workers than among those of professional and business people.

The basic cause of a stillbirth may be very different from the immediate cause. For example, stillbirth from intracranial haemorrhage may be caused by over-moulding of the head in its passage through a small pelvis. Although the immediate cause of death is intracranial haemorrhage the basic cause is pelvic contraction, which in turn may have developed because the mother's environment in childhood was faulty and a deficient diet stunted her skeleton. It seems reasonable to postulate that such a mother will show other effects of reproductive inefficiency, which a high standard of antenatal care may be unable to avert. If this should be the case then, as Dugald Baird (1955) has said, the moral is clear that the planning of a pregnancy should begin when the mother herself is born.

Stillbirth rates between 1929 and 1939 varied but little, but between 1939 and 1945 a most dramatic decline in the incidence of stillbirths took place. This is illustrated by the graphs in Fig. 1 (after Ryle, 1948):

The decline in the number of stillbirths began during World War II. This is all the more remarkable when one recalls what living conditions were like during that time. There appear to be several reasons to account for this improvement. The first is that, with the introduction of rationing of food, pregnant mothers were ensured of a diet that contained the essential elements and foodstuffs. Then there were well-organized antenatal clinics made available in the areas to which the pregnant mothers had been evacuated and, in the spirit of the times, these pregnant women attended them almost as a national effort. Blood-transfusion services became highly organized and penicillin came into use, both of which must have had an indirect but beneficial effect. Lastly, but not least, a change seems to have come into obstetric practice, inasmuch as the more heroic measures to deliver vaginally were abandoned in favour of delivery by the lower-segment Caesarean section, which operation became very much safer with blood transfusions and antibiotics at hand.

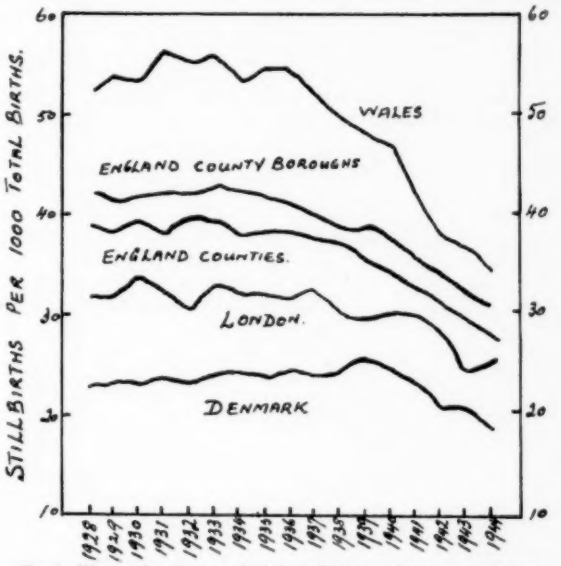

Fig. 1. Illustrating the trend of the stillbirth rate in geographical areas of England and Wales and in Denmark (1928 to 1944).

QUEEN VICTORIA HOSPITAL STATISTICS

The stillbirth rate and the neonatal death rate for the Queen Victoria Hospital, Johannesburg, for the years 1952, 1953 and 1954 will now be considered and this

* A paper presented at the South African Medical Congress, Pretoria, October 1955.

will be followed by a brief analysis of the stillbirths and the neonatal deaths for these 3 years.

The total number of deliveries was 3,866 in 1952, 3,913 in 1953, and 3,988 in 1954. These totals (11,767 in the 3 years) include all deliveries, booked and unbooked, 'hospital' and 'district'.

Stillbirth Rates

The total uncorrected stillbirth rates for the 3 years are as follows:

1952—21·9 per 1,000 births
1953—19·1 per 1,000 births
1954—15·8 per 1,000 births

Now, if only the patients who attended the antenatal clinic are considered—and this group forms 94% of our total practice—the uncorrected stillbirth rates are as follows:

1952—17·1 per 1,000 births
1953—13 per 1,000 births
1954—11·9 per 1,000 births

Correcting these latter figures for macerated stillbirths and for gross congenital abnormalities, then the corrected stillbirth rates, for antenatal-clinic cases are:

1952—6·5 per 1,000 births
1953—3·9 per 1,000 births
1954—2·9 per 1,000 births

There has thus been a progressive improvement over the 3 years. This improvement may partly be explained by stricter and more diligent supervision in the antenatal wards and in the labour wards of the hospital, and also by the freer use of Caesarean section in the treatment of placenta praevia and also in those cases where foetal distress occurred in the first stage of labour. The uncorrected stillbirth rate for all antenatal-clinic patients delivered in 1954—11·9 per 1,000 births—is getting close to the 'biological possible' of 10 per 1,000 mentioned by Baird (1947).

Neonatal Death Rates

The neonatal death rate has remained practically the same over the 3 years. The uncorrected neonatal death rates for all cases are as follows:

1952—17·2 per 1,000 live births
1953—16·1 per 1,000 live births
1954—19·8 per 1,000 live births

The uncorrected neonatal death rates for patients attending the antenatal clinic are:

1952—14·4 per 1,000 live births
1953—12·5 per 1,000 live births
1954—12·2 per 1,000 live births

If these figures are corrected for all infants of less than 32 weeks, for those with gross congenital abnormalities and for the cases of hydrops foetalis, then the corrected neonatal death rates for antenatal clinic patients become:

1952—7·8 per 1,000 live births
1953—8·0 per 1,000 live births
1954—7·9 per 1,000 live births

Though the freer use of Caesarean section has reduced the incidence of stillbirths in cases of placenta praevia, nevertheless a proportion of these infants are lost in the neonatal period from prematurity and its associated conditions.

For the sake of clarity the analysis of our perinatal mortality will be presented in the 3 natural divisions: macerated stillbirths, fresh stillbirths and neonatal deaths.

MACERATED STILLBIRTHS

There were 117 macerated stillbirths born in the 3 years 1952, 1953 and 1954. In the great majority of these cases death had occurred *in utero* well before the onset of labour. In other words, the intra-uterine death had no relationship to the actual labour or method of delivery.

A striking feature is revealed when the ages of the mothers are considered. Comparing the ages of these mothers with the ages of all the mothers delivered in the hospital, it becomes clear that macerated stillbirths occur much less frequently among young mothers and much more frequently among mothers over the age of 30 years:

Age of Mother	*Macerated Foetus*	*All Deliveries*
Under 20	5·9%	14·5%
20-29	47·9%	62·0%
30-39	39·4%	20·7%
40 and over	6·8%	2·8%

Thus the chance of a mother under 20 years of age producing a macerated stillbirth is less than half that of the average mother. Similarly, macerated stillbirths occurred twice as commonly in the mother over 30 years of age. It follows then that an age factor must either directly or indirectly play an important part in the occurrence of macerated stillbirths.

When parity is considered, it is seen that there are twice as many multiparae as primiparae. Comparing parity in this group with parity for all cases delivered in the hospital, it becomes obvious that macerated stillbirths occur less frequently among primiparae:

	Macerated Stillbirths	*All Deliveries*
Primiparae	31·6%	46·5%
Multiparae	68·4%	53·5%

No doubt the age factor mentioned above must play some part in explaining this preponderance of multiparae over primiparae.

It was found that 60% of the macerated foetuses weighed less than 5½ lb. at birth, which means that by the international standard for prematurity these 60% were premature, and that the factor causing intra-uterine death must have been operative in the majority of the cases before the 36th week of pregnancy.

The causes of the 117 macerated stillbirths of this series are expressed briefly by the following table:

Cause	*Number*	*Percentage*
Cause unknown	57	48·7%
Toxaemia of pregnancy ..	17	14·5%
Placenta grossly infarcted (? pre-eclampsia)	10	8·5%

Cause	*Number*	*Percentage*
Accidental haemorrhage (? pre-eclampsia)	5	4·3%
Chronic nephritis	1	0·9%
Diabetes *plus* pre-eclampsia	1	0·9%
Rh antibodies	15	12·8%
Accidents to the cord (cord tightly around neck, true knots, etc).	7	6·0%
Gross congenital abnormalities	3	2·6%
Extra-uterine pregnancy	1	0·9%

The first point that strikes one from this analysis is that in about one half of the cases the cause of the stillbirth is unexplained. This finding is in accordance with other analyses of macerated stillbirths found in the literature. It is in these mysterious deaths that a large field for further research lies. The age of the mother, her hereditary factors, her diet, her endocrine system must all be investigated, and then, of course, the placenta itself must come under strong suspicion. One hopes that further investigations into placental metabolism will eventually throw some light on these possibly preventible deaths.

Toxaemia of pregnancy accounts for the next largest group of macerated stillbirths (14·5%). In a further 8½% of the cases a grossly infarcted placenta was found—and possibly some of these at least might have been associated with a pre-eclampsia whose signs disappeared after the death of the foetus. Another 4·3% were associated with accidental haemorrhage, and one cannot but feel that there must have been an underlying toxaemic factor in some of these. Chronic nephritis and diabetes complicated by toxaemia of pregnancy each accounted for a further 0·9% of the cases. These cases have been placed together because of the common background of toxaemia of pregnancy, which must be implicated in the causation of these intra-uterine deaths. Possibly earlier diagnosis and treatment of the toxaemia might have prevented some of these deaths.

The presence of the Rh antibodies accounted for as many as 12·8% of the macerated stillbirths. In the present state of our knowledge regarding the Rh antibodies those deaths that occur from this cause before the 36th or 37th week of pregnancy are unavoidable.

No less than 6% of the intra-uterine deaths were caused by mechanical obstruction of the blood flow in the umbilical cord, by true knots of the cord or because the cord was tightly around the foetal neck or body. One feels that these too are unavoidable.

The 2·6% caused by gross congenital abnormalities may have been due to hereditary factors, or environmental and dietetic factors. It has been shown that in animals certain deficient diets given to the mother will result in abnormal offspring: whether this is so in human beings as well is yet to be demonstrated.

Advanced extra-uterine pregnancy, which accounted for 1 case of macerated stillbirth, is fortunately very rarely met with.

FRESH STILLBIRTHS

There was a total of 97 fresh stillbirths in the 3 years under consideration. The fact that they were fresh indicates that the death occurred within 24 hours of delivery, and so it follows that the majority of these deaths occurred during labour and were associated in some way with labour or delivery.

An analysis of the ages of the mother again shows a greater incidence of fresh stillbirths in those over the age of 30 years. In those over 40 the incidence is more than 5 times the average:

Age of Mother	*Fresh Stillbirths*	*All Deliveries*
Under 20	11·4%	14·5%
20-29	39·6%	61·98%
30-39	33·4%	20·66%
40 and over	15·6%	2·8%

Both the unknown age-factor and the factors involved with grande multiparity must enter into this very much higher stillbirth rate over the age of 40 years. In our series fresh stillbirths were found to occur relatively less frequently in primiparae than in multiparae:

	Fresh Stillbirths	*All Deliveries*
Primiparae	33·3%	46·5%
Multiparae	66·6%	53·5%

This can largely be explained by the fact that the hospital admits all primiparae, but tries to limit admission of multiparae to those who are, or have been, abnormal obstetrically. A further analysis into the degrees of parity would probably show, as other analyses have shown, that the stillbirth rate is higher in primiparae than in multiparae who have had 1, 2, 3 or 4 children, but that after the 5th delivery the stillbirth rate rises above that found in primiparae.

Unlike the macerated stillbirths, the majority of fresh stillbirths were over 5½ pounds at birth:

	Macerated Stillbirths	*Fresh Stillbirths*
Less than 5½ lb.	60%	35%
Over 5½ lb.	40%	65%

The method of delivery of the 97 fresh stillbirths was as follows:

	Number	*Percentage*
Spontaneous vertex delivery	54	55·8%
Breech delivery	14	14·4%
Internal version and breech extraction	10	10·3%
Craniotomy and extraction	5	5·1%
Forceps delivery	8	8·2%
Caesarean section	3	3·1%
Laparotomy (ruptured uterus)	3	3·1%

Of the 5 craniotomies, 3 were done for hydrocephalus and the other 2 for obstructed labour where the foetal heart sounds had disappeared. Of the 8 forceps deliveries 6 were done for marked foetal distress in labour, and the other 2 in severely toxaemic patients where the foetal heartsounds had disappeared. Of the 3 Caesarean sections 1 was done in a patient with type-4 placenta praevia in whom no foetal heart could be heard on admission, and the other 2 for foetal distress in early labour.

The causes of fresh stillbirth make a long and varied

list and it may be profitable to group them under 3 general headings:

1. CONDITIONS ASSOCIATED WITH PREGNANCY

	Number	Percentage
Toxaemia of pregnancy ..	11	11·3%
Accidental haemorrhage ..	15	15·5%
Placental praevia	7	7·2%
Hydrops foetalis	3	3·1%
Chronic nephritis	1	
Syphilis	1	
External version followed by retroplacental haematoma ..	1	4·1%
Premature rupture of membranes (5 days before onset of labour)	1	
	40	41·2%

Of the cases caused by toxaemia of pregnancy 3 were associated with eclampsia and the other 8 with a severe form of pre-eclampsia, half this number occurring between the 28th and 34th week of pregnancy. Of the cases that succumbed to accidental haemorrhage 2/3rds were also associated with severe pre-eclampsia. Of the 7 fresh stillbirths associated with placenta praevia 1 was delivered by Caesarean section for type-4 placenta praevia, in spite of there being no foetal heart sounds on admission; the other 6 were delivered vaginally. The stillbirth due to congenital syphilis occurred at the 34th week of pregnancy in a patient who had only just commenced treatment and who had a history of a previous stillbirth probably due to the same cause.

2. CONDITIONS ASSOCIATED WITH LABOUR

	Number	Percentage
Accidents to the cord (prolapse, cord around neck, etc) ..	11	11·3%
Aftercoming head held by cervix (premature breech)	4	4·1%
Foetal distress in labour with disappearance of the foetal heart	8	8·2%
Internal version for transverse lie (also with foetal distress)	4	4·1%
Cerebral haemorrhage (1 forceps, 1 brow)	2	2·1%
Ruptured uterus	3	3·1%
	32	32·9%

Most of the cases of accidents to the cord, which accounted for 11·3% of the fresh stillbirths of this series, were sent into the hospital because of these conditions; in a large proportion of them the foetal heart-beat had already disappeared on admission. Prolapse of the cord was usually associated with premature labour or with abnormal presentations. The 8 fresh stillbirths associated with foetal distress in labour were theoretically avoidable if prompt delivery had been effected. Of the 3 ruptured uteri 2 were ruptures through a previous classical Caesarean-section scar, and the 3rd was associated with a brow presentation. These are also avoidable tragedies—provided the case is seen in time.

3. MISCELLANEOUS

	Number	Percentage
Cause unknown	15	15·5%
Gross congenital abnormalities	10	10·3%
	25	25·8%

The group of 'unknown causes' were the cases admitted in labour in which no foetal heart could be heard on admission, and in whom no obvious cause for the stillbirth could be found. In all these the placenta came under suspicion, but macroscopically they appeared quite normal. Nevertheless, one feels that 'placental insufficiency' must account for some of these. The group of major congenital abnormalities is a large one and with the improvement in the incidence of stillbirths due to other causes, it is assuming greater prominence as a cause of stillbirth.

NEONATAL DEATHS

During the 3 years 1952-54 there were 206 neonatal deaths among all cases admitted to the hospital. This number includes 9 pairs of twins. It also includes 19 cases admitted to hospital after delivery outside, mostly because of premature infants. The age of the mother does not appear to have the same significance here as it does with stillbirths, except that once again mothers over the age of 40 years are associated with a slightly higher incidence of neonatal deaths.

The duration of pregnancy in weeks at the time of delivery of the 206 infants is as follows:

28-32 weeks .. 42·2%
32-36 weeks .. 28·7%
36 weeks and over 29·1%

It is no surprise to find the largest proportion of neonatal deaths amongst those infants who were less than 32 weeks. If prematurity is gauged by the international standard of 5½ lb. birth-weight and less, then we find that 75% of the neonatal deaths fall into this group (155 out of 206).

There was a proponderance of male infants in the group of neonatal deaths, 56·3% of the infants being male and only 43·7% being female.

No less than 60% of the neonatal deaths occurred within the first 24 hours of life.

The method of delivery has been considered in two sections: (1) infants less than 5½lb. and (2) infants over 5½lb.

1. METHOD OF DELIVERY OF INFANTS 5½ LB AND LESS

Method	Number	Percentage
Spontaneous vertex delivery ..	110	71%
Breech delivery	27	17·4%
Internal version	2	1·3%
Forceps delivery	3	1·9%
Caesarean section	13	8·4%
	155	100%

Of the 13 Caesarean sections, 6 were done for placenta praevia, 5 for severe pre-eclampsia, 1 for chronic nephritis, and 1 for carcinoma of the breast.

2. METHOD OF DELIVERY OF INFANTS OF 5½ POUNDS AND OVER

Method	Number	Percentage
Spontaneous vertex delivery ..	43	84·3%
Breech delivery	3	5·9%
Internal version	0	0
Forceps delivery	2	3·9%
Caesarean section	3	5·9%
	51	100%

Of the Caesarean sections 2 were done for diabetes, and 1 for foetal distress in the first stage of labour.

The causes of the 206 neonatal deaths is presented most briefly in the form of a table:

Cause	*Number*	*Percentage*
Prematurity	96	46·6%
Prematurity associated with pre-eclampsia	10	4·9%
Prematurity associated with diabetes	3	1·5%
Atelectasis	39	19·0%
Bronchopneumonia	4	2·0%
Cerebral haemorrhage	18	8·7%
Rh antibodies	9	4·4%
Gross congenital abnormalities	22	10·9%
Congenital syphilis	1	
Cerebral oedema and adrenal haemorrhage	1	2·0%
Gastro-intestinal haemorrhage	1	
Cause unknown (no cause of death found P.M.)	1	

The largest group falls under 'prematurity', and though immaturity of the infant must be the usual cause of the death, there were probably a good number in whom atelectasis and the hyaline-membrane syndrome were the factors which actually precipitated death. Of the 18 cases of cerebral haemorrhage, 1 was delivered by forceps and 1 by Caesarean section; the remaining 16 were spontaneous deliveries. No doubt some of these, cerebral haemorrhages were caused by an asphyxial state during the delivery.

CONCLUSION

Perinatal mortality therefore, is a challenge which must be met in a diversity of ways. Basically it is associated with the health of the nation, particularly the standard of nutrition and the general standard of society. More specifically it is concerned with expert antenatal supervision and with adequate facilities and organization for recognizing any abnormality early and dealing correctly with it. In Nature's way of things, the perinatal mortality will never be completely eliminated, but the constant striving towards this ideal should bring out all that is best in obstetric practice.

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CHOLEDOCHAL CYST

SAMUEL SKAPINKER, M.B., B.CH. (RAND), F.R.C.S. (EDIN.)

Department of Surgery, University of Witwatersrand, and Johannesburg General Hospital, Johannesburg

The first reported case of choledochal cyst was described by Vater 230 years ago. In 1946 Shallon, Eger and Wagner[1] reviewed the literature. In 1955 Alton and Obeid[2] added 17 more cases, making the total reported cases 201. Fleming[3] in 1947 reported a case that occured in South Africa. In the records of the Mayo Clinic, Everett Shocket *et al.*[4] found 6 cases to add to the records.

A choledochal cyst is an aneurysmal dilatation of a segment of the biliary tract. It is a disease much more prevalent in women than in men and an analysis of the above cases shows it to be 3 times commoner in females than in males. The usual age of onset is under 25 years, but many cases wait several years before surgical treatment is undertaken.

The origin of the cysts is unknown, but generally it is accepted that they are congenital, because the majority occur in children and the incidence diminishes with age. Cysts have been described in an unborn child,[5] as well as in newborn children.[6, 7, 8] Another factor supporting the congenital origin of these cysts, is that they have been described associated with other congenital abnormalities of the biliary tract and liver. Gross[9] advances the theory that there is a congenital weakness of the duct associated with a valve of the lower end causing obstruction. The congenital cyst usually involves the supraduodenal portion of the common bile-duct, and is confined to the common bile-duct. There is no associated dilatation in the hepatic duct. The cyst varies in size and may reach an enormous magnitude. Reel and Burrell[10] reported a case which contained 8 litres. The cyst wall is made up of fibrous tissue.

Symptomatology and Diagnosis

The commonest symptom-traid is a right upper abdominal tumour associated with jaundice and pain. The mass is usually large, cystic and easily palpable. Jaundice is found in 3 out of every 4 cases and is usually of the obstructive type. Pain may be slight and usually there is a fairly long history. The nature of the pain may be colicky, or it may be due to pressure on the surrounding structures, causing a 'dragging' sensation.

The mass is commonly misdiagnosed as a pancreatic cyst. Only about 50% of the cases are correctly diagnosed pre-operatively. The diagnosis may be made with this symptom-triad and X-ray studies of the case. Barium meal usually shows the duodenum pushed outwards, and forwards as can be seen demonstrated in the X-rays of the patient now described. Pancreatic cysts usually push the duodenum outwards and backwards. Cholecystogram, if concentration occurs, usually reveals the gall-bladder pushed upwards and indented, giving a comma shape. Alton and Obeid,[2] however state, that both the gall-bladder and the choledochal cyst do not concentrate even when intravenous Biligrafin is used.[2]

Treatment

Since the diagnosis is commonly not made pre-operatively, complications may occur if an attempt is made to resect the cyst without realizing what it is. The

safest procedure is to aspirate the cyst when, if bile is obtained, the condition should be recognized and a small opening is made into the sac. The contents are then aspirated and the communications to the ducts can be demonstrated. If the opening to the duodenum cannot be seen, this does not preclude the diagnosis.

Excision of the cyst is an extremely difficult and dangerous procedure and should not be embarked on, because of the high mortality. The procedure of choice is a short-circuiting operation. Alton and Obeid[2] reviewed 210 cases and found that 22 were not operated on, all of which died. The cause of death was rupture of the cyst, suppurative cholangitis and cirrhosis of the liver. The child will suffer recurrent attacks of pain and jaundice. Anastomosis with the duodenum results in a 24% mortality due to shock, ascending cholangitis, bleeding or liver failure. Roux Y anastomosis of the cyst with the jejunum would appear to be the treatment of choice, for the possibility of ascending cholangitis is avoided. The case now described is the 11th of all the collected series in which this procedure was carried out; all these cases survived.

CASE REPORT

Mrs A.M.P., a housewife aged 39, complained of jaundice and a mass in the epigastrium. She was an extremely poor witness and stated that this mass and jaundice suddenly appeared 2 weeks previously. Pain had been intermittent over the same period. There was nothing significant in her past history except that she was an alcoholic.

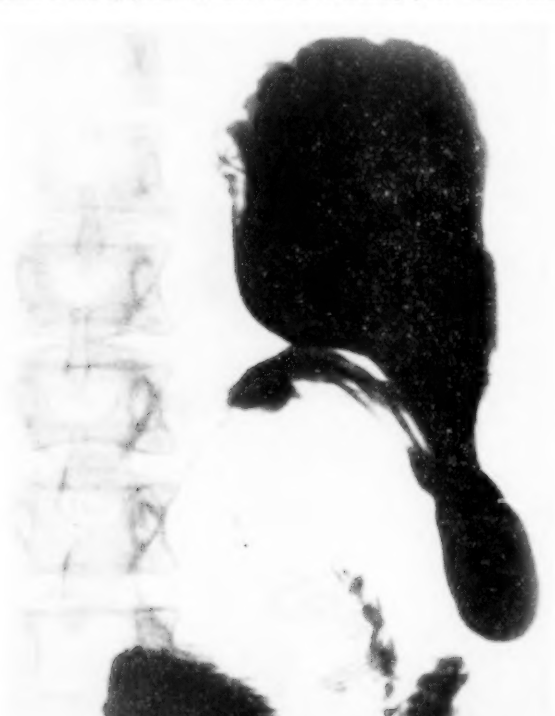

Fig. 1. Showing the 'C' curve of the duodenum greatly enlarged and the descending loop of the duodenum depressed.

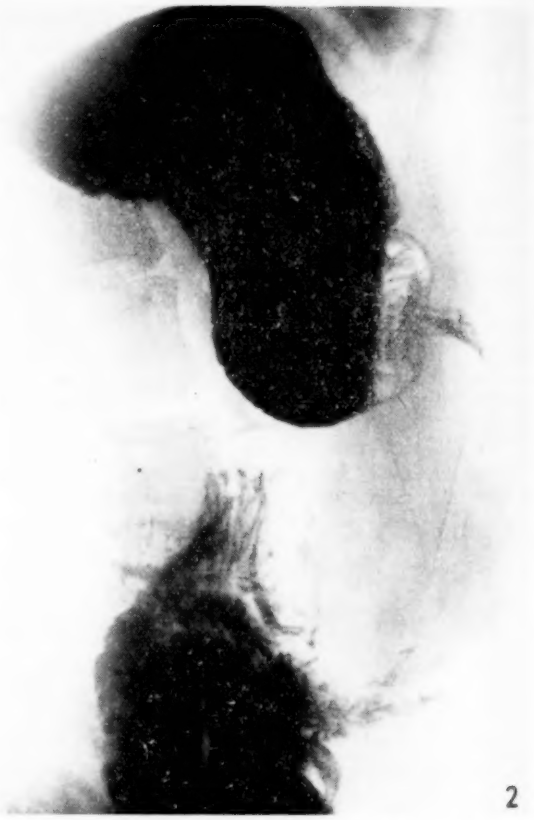

Fig. 2. The duodenum is displaced markedly forward by the choledochal cyst.

She was a thin woman, deeply jaundiced. Abdominal examination revealed a liver that was 3 finger-breaths enlarged below the costal margin and there was a large smooth cystic mass below it. The mass was 8 inches in diameter and was freely mobile, especially in the horizontal plane. Liver function and blood tests were performed with the following results (Dr. I. Bersohn, 11 August, 1955):

Hb. 14·2%. Red cells 4,840,000 per c.mm. Leucocytes 10,400 per c.mm. (neutrophils 69·5%, monocytes 4·5%, lymphocytes 23·0%, eosonophils 3·0%).

The red cells show slight anisocytosis and a tendency towards hypochromasia.

Platelets were present in normal numbers. Occasional target cells were seen.

ESR 33 mm. in 1 hour.

PCV 43·5.

PI 89% (control clotted in 11·0 sec., specimen clotted in 12·4 sec.).

Bilirubin++++., Urobilin++++., Urobilinogen present, Wallace Diamond dil. 1/30.

Thymol turbidity 1·0 unit, Thymol flocculation negative.

Colloidal-red test—negative.

Cephalin cholesterol flocculation+++.

Takata Ara reaction—negative.

Zinc sulphate turbidity—15·8 units.

Total lipid—934 mg.

Alkaline phosphatase—90 units.
Van den Bergh—prompt direct.
Bilirubin direct—3·2 mg., bilirubin total—4·6 mg.
Total protein—6·9 g. %, albumin—3·3 g. %, globulin—3·6 g. %, gamma globulin—1·54 g. %.
Serum cholinesterase—69% of the average normal activity.
Mucoprotein—163 mg. %.
Polysaccharide of mucoprotein—29 mg. %.
P/M Ratio—18.

X-rays (Dr. E. Samuel) taken on the same date revealed that the stomach was displaced towards the left side and a large mass occupied the lesser sac, displacing the stomach forwards and towards the left (Fig. 1). The 'C' curve of the duodenum was greatly enlarged and the descending loop of the duodenum was depressed and markedly displaced forwards (Fig. 2). Cholangiogram revealed a virtual obstruction of the common duct.

Operation was performed on 15 August 1954. A transverse upper abdominal incision was made and, when the peritoneum, was opened, a large mobile cyst was found pushing the duodenum forward. The duodenum was markedly stretched. The gall-bladder and common hepatic duct were markedly dilated. The gastro-colic omentum was divided and the cyst shelled easily to its attachment to the common duct. The cyst was then punctured and 20 oz. of bile evacuated. The diagnosis being made, a Roux Y anastomosis was performed 12 inches from the duodeno-jejunal flexure, and a proximal loop of 10 inches was brought up and anastomosed to the cyst.

The patient made an uneventful recovery.

The biliary fluid was sent for examination and tests for bile pigments were positive. On spectroscopic examination, methaemoglobin was detected. Biopsy of the wall of the cyst was also taken and this revealed chronically inflamed connective tissue lined on the one side by granulation tissue.

SUMMARY

1. A review of the literature on congenital cysts of the bile ducts is presented.

2. Anastomosing the cyst with the jejunum by means of the Roux Y procedure is suggested as the method of choice.

3. The dangers of excision and marsupulization of the sac are shock and ascending cholangitis.

4. An additional case of choledochal cyst is described.

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AN OPHTHALMOLOGICAL SURVEY OF A SERIES OF CEREBRAL PALSY CASES

L. SCHRIRE, M.Sc., M.B., CH.B., D.O.M.S.

Kimberley

Cerebral palsy (in this paper denominated CP) has been defined by Phelps[1] as 'a group of conditions which affect the control of the voluntary motor system and which have their origin in lesions of various parts of the brain.' The condition is classified according to the situation of the lesion. Three main groups into which 95% of the cases fall, are recognized:

1. *Spastic.* The lesion is in the cerebral cortex or pyramidal tract.

2. *Athetotic* (dyskinesic—Breakey[10]). The lesion is in the basal ganglia.

3. *Ataxic.* The lesion is in the cerebellum.

There are 3 other rare types, namely the rigid, the flaccid and the tremor types.

CP was first described by Little, in his communications of 1853 and 1862. His concept of a natal aetiology of the condition was challenged later by McNutt (1885), Osler (1889), Sigmund Freud (1897), and many investigators in the 20th century (see Anderson[2]).

Before 1939, treatment was directed mostly to operations performed on the most affected muscle-groups of the limbs but, as a result of the pioneer work of Phelps, Carlson and Perlstein, the line of treatment has swung to treating the child as a whole, with team-work by the parent, teacher, family doctor, medical specialists, physiotherapist, occupational therapist, etc. Special clinics, hospitals and institutions have been set up, notably in the USA. In South Africa, too, there are several institutions which cater for these cases.

AETIOLOGY

The condition is for the most part unavoidable and a decrease of its frequency in the near future is not to be expected. Evans[3, 11] pointed out, however, that if our present knowledge was applied the incidence might be reduced by 1/3rd. The aetiological factors fall into 3 chronological periods (Phelps[1]):

1. *Prenatal*

(*a*) Congenital normal variations in the size, shape and functions of the brain.

(*b*) Congenital defective development (interference at any state of pregnancy, independent of heredity or specific prenatal environmental influences).

(*c*) Pathological prenatal conditions (e.g. vitamin or calcium deficiencies, disturbances of endocrine glands, liver or kidney, etc.)

2. *Natal*

Birth trauma, e.g. prematurity with rapid birth, forceps delivery, anoxaemia, Rh-negative mothers, etc.

('it is impossible to blame "poor obstetrics" for more than 3% of birth-injured CPs.'[4])

3. *Postnatal*

(*a*) Convulsions leading to cerebral haemorrhage during the first 3 months of life.
(*b*) Pertussis under 6 months.
(*c*) Encephalitis.
(*d*) Head trauma, e.g. falls.
(*e*) In adults, cerebral accidents.

INCIDENCE

Phelps[1] has stated that in every 100,000 of the population 7 CP cases occur. Of these one dies under the age of 6; 2 are feeble-minded and require custodial care; and, of the remaining 4 who are mentally normal, one is severely handicapped, requiring custodial care and education, 2 are moderately affected and can be rehabilitated by treatment, and 1 is so mild that treatment is unnecessary. Phelps estimated therefore that in the USA there is a total of 200,000 cases up to the age of 20; i.e. CP is second to poliomyelitis as a child crippler. Levin, Brightman and Burtt[5] give much higher figures in their survey of Schenectady county.

Asher and Schonell[6] considered the incidence of CP in the school-going population as 1 in every 1,000.

In South Africa, no estimate can reliably be made. This is partly due to the fact that such various grades of cerebral damage are possible, partly because of the relatively light concentration of doctors in the country areas, where the Native population through ignorance and superstition, economic or spacial reasons, seldom is able or willing to consult a qualified medical practitioner. Medalie[7] has estimated an incidence of approximately 1,000 white children in the White population of 3,000,000.

Considering the population of South Africa as being over 12 millions, (12,646,375, the latest preliminary census figure) there should therefore be approximately 4,000 CP cases in the country.

OPHTHALMIC INVESTIGATIONS

Very little has been written in the ophthalmic literature on the subject of CP. The main contributors so far have been Guibor[8, 9] and Breakey[10]. As I have been fortunate enough to see a number of cases of this disability in my private practice, it was thought that a report might be of interest. These cases were drawn from a local school where school-going girls and boys from about the age of 5½ upwards are treated.

Material and Methods

A total of 73 unselected cases were examined. Some of these have been under my observation for 4 or 5 years. Table I shows them classified according to age, sex and type of case. It will be noted that 63 (or 86%) are spastic in nature. The sex relationship is approximately 2 males to 1 female (48 : 25); no valid conclusion can be drawn from this, as it may be merely due to the greater availability of accommodation for the male school children. The eye examination consisted of:

1. Visual acuity determination on the Snellan's chart or illiterate 'E' chart at a measured 6 metres; refraction with or without mydriatics (4% homatropine cocaine or atropine 1%) as indicated; subjective correction with lenses.

2. Muscle-balance studies with the cover test, the Maddox wing for near, and the Maddox rod with separate prisms or the Risley rotary prism for distance.

3. General eye examination.

TABLE I. ANALYSIS OF TYPE OF CP ACCORDING TO SEX AND AGE

	5—9		10—14		15—20		Over 20		
	M	*F*	*M*	*F*	*M*	*F*	*M*	*F*	*Total*
Spastic	7	6	13	3	21	12	—	1	63
Athetoid	2	—	2	—	2	1	—	—	7
Ataxic	—	1	1	—	—	1	—	—	3
Total	9	7	16	3	23	14	—	1	73

RESULTS

The findings were considered under 3 headings: (1) Refraction, (2) muscle balance, and (3) other associated neural or neuromuscular defects.

1. *The Status of the Refraction*

As emmetropia, though the ideal optical condition is biologically unusual, it was arbitrarily decided to use the term 'normal' where the patient was ocularly symptom-free and the range of the refractive state lay between + 1·00D.sph. and —0·50 D. sph. and between +0·50 D. cyl and —0·50 D. cyl. (axis immaterial). Table II reflects the findings in the various types of CP when classified under the headings of 'normal', more hypermetropic than + 1·00 D. sph., more myopic than —0·50 D. sph., and more than 0·50 D. of hypermetropic astigmatism (compound myopic astigmatism was classed as myopia). It is to be noted that 54 (or 80%) can be considered 'normal'. There were 12 cases of amblyopia, mostly associated with strabismus or anisometropia. For various reasons it was impossible to test the refraction of 5 of the more spastic children.

TABLE II. THE REFRACTIVE STATUS ANALYSED WITH REFERENCE TO THE TYPE OF CEREBRAL PALSY

Classification	*Spastic*	*Athetotic*	*Ataxic*	*Total*
'Normal'	47	5	2	54
More hypermetropic than + 1·00 D. Sph.	1	1	—	2
More myopic than —0·50 D.Sph. ..	8	—	—	8
More than —0·50 D. Cyl. hypermetropic astigmatism	3	1	—	4

It was impossible for various reasons to test the refraction of 5 of the cases

2. *Muscle Balance*

The patient was considered to have a phoria (once more arbitrarily) if he had more than 4 prism-dioptres

TABLE III. THE PHORIAS AND TROPIAS ANALYSED ACCORDING TO TYPE OF CEREBRAL PALSY

	Spastic	*Athetotic*	*Ataxic*	*Total*
Heterophoria ..	9	3	—	12
Esotropia ..	7	—	1	8
Exotropia ..	3	—	—	3

of exo- or esophoria or 1 prism-dioptre of hyperphoria. The patient was considered to have strabismus if it was either constant or periodic at the time of examination. It was noted that in this series there were 12 cases of heterophoria, 8 cases of convergent strabismus and 3 cases.

TABLE IV. INCIDENCE OF OCULAR DEFECTS WITH CEREBRAL PALSY COMPARISON WITH RESULTS OF PREVIOUS INVESTIGATORS. PERCENTAGES.

	Guibor	*Breakey*	*This Series*
Amblyopia	25		16
Esotropia	51	40	11
Exotropia	9	8	4
Horizontal Conjugate Defects	33		
Ptosis	2	—	—
Spastic Lids		1	—
External Ophthalmoplegia			1
Nystagmus	9	2	4
Coloboma of Iris		1	—
Congenital Cataract		2	—
Papilloedema		1	—
Optic Atrophy	2	3	4
Previous Choroiditis			1

of divergent strabismus. Table III demonstrates these findings. It can be deduced from these results that there is a greater number of muscle defects in these CP cases than in the general population, a view held by previous investigators.

3. *Other Associated Abnormalities*

In this series, the following associated abnormalities were noted:

Optic atrophy—3 cases (4·1%).
Total external ophthalmoplegia—1 case.
Nystagmus—3 cases (4·1%).
Old healed choroiditis—1 case*.
Anisocoria—1 case.
Homonymous hemianopia—1 case.

* This case was fully investigated clinically and radiologically (including an examination for toxoplasmosis) with negative results.

Table IV is a comparison of the findings of this series with those of Guibor and Breakey.

DISCUSSION

It has been shown previously that the incidence, particularly of squints, is higher in the CP patients than in the general population. Guibor[8] concluded that 75% of his CP cases had motor defects. He states that this percentage is probably higher than the statistics usually found. His patients appear to have been mostly seen below 2 years of age. The present results show a lower percentage of abnormalities possibly because (1) the cases belonged to an older age-group than Guibor's, and (2) severe CP cases were not seen. The cases seen could be considered as a selected group because, firstly, all cases were White (Europeans) and, secondly, a requisite for their admissibility into the local schools was that they were 'educable'. In other words they belonged to the rehabilitable 2/7ths of Phelps' classification. It is felt that severer signs would be demonstrated in cases with more severely damaged brains.

SUMMARY

1. A total of 73 cases of cerebral palsy were examined ophthalmologically.

2. A larger percentage of squints and other neuromuscular lesions were demonstrated than would be expected in a similar sample of the general population. This bears out the conclusions of previous investigators.

I should like to thank Drs. M. M. Pretorius and E. du Plessis for referring cases and giving me facilities which made this survey possible.

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SOME OBSTETRICAL PROBLEMS ENCOUNTERED IN GENERAL PRACTICE*

A. ROSIN, M.B., CH.B. (CAPE TOWN)

Queenstown

False Labour Pains

Not infrequently cases are referred to hospital, or the obstetrician is called into consultation, because in spite of severe uterine pains there is no progress in labour. Many of these cases are women with false labour pains. These women are often told some fantastic tale about why the baby cannot descend and that the only hope for mother and baby is a Ceasarean section.

Painful uterine contractions do not necessarily mean that labour has commenced. When these patients are examined it is noticed that the force of contractions is out of all proportion to the pain. False labour pains may vary in intensity from niggling discomfort to severe pain.

The important fact which is forgotten is that dilatation of the cervix is the only true indication that labour is in progress. If a rectal examination or, under suitable circumstances, a vaginal examination is done, the doctor will save himself unnecessary journeys because of a so-called obstructed labour. I can recall very vividly being called into the country to see just such a case. The nervous, worried mother and relatives had already been told that a Caesarean section was imperative. Examination showed that the woman was obviously having false labour pains. Unfortunately, the practitioner would not yield and insisted on a

* A paper presented at the South African Medical Congress, Pretoria, October 1955.

Caesarean section. Ours is a profession where honesty and integrity are of first importance. The doctor must not be afraid to 'lose face'.

Pelvic Assessment

Two cases of vesico-vaginal fistula recently seen were both associated with 'failed forceps'. If some attempt at a pelvic assessment had been made, in neither case would the hopelessly contracted funnel character of the pelvis have been missed. There are senior practitioners who are unaware of the value of pelvic assessment, and some junior men are so ready to apply forceps under inadequate supervision that the simple routine examinations for application of forceps are forgotten, with disastrous results both for mother and child.

Where the foetal head has entered the pelvis and obstruction has occurred, it is as well to remember the possibility of funnelling of the pelvis with a small sub-pubic angle and a narrow bi-ischial diameter.

Is the patient at term?

Cases are frequently seen going 1, 2 or even 3 weeks past term, and the question arises, is the case truly post-mature? It is extraordinary how very unobservant some women are about their monthly physiological processes, but careful questioning about the last menstrual period will very often help in coming to a more satisfactory conclusion about the due date.

Another very important point is the state of the cervix. Cases are frequently referred because the woman is supposed to be due and on vaginal examination a long, uneffaced, undilated cervix is found. Such women cannot be at term and may be sent home.

Premature Rupture of Membranes

Spontaneous rupture of the membranes 2 or 3 weeks from term without the patient's going into labour is a problem which occasionally confronts the practitioner. In these cases the following procedure should be adopted:

1. Examine the vulva for any obvious sign of cord prolapse. Listen for the foetal heart.
2. Avoid doing vaginal examinations. A woman will seldom go septic with spontaneous rupture of membranes, but once fingers are introduced into the vagina the risk of sepsis is considerable.
3. Use medical induction, giving castor oil, hot shower and enema, and repeat, if necessary, 36 hours later.
4. Order the bed linen to be changed completely every 24 hours.

These patients, even if they do not go into labour, seem to come to no harm.

Premature Labour

The chief danger of premature labour to the baby is cerebral haemorrhage. The premature infant has a low prothrombin blood-level. Its cerebral tissues cannot take the strain of the birth process. I give 10 mg. of vitamin K by intravenous injection to the mother.

In these cases the premature infant will easily traverse the pelvis, but catastrophe occurs at the perineum. Rather than watch the foetal head straining at the rigid perineum of the primipara, it is far better to do an early episiotomy. The doctor will not usually hesitate to do an episiotomy for a full-term baby, but for some unknown reason he always feels that the head of the premature infant is too small to be held back by the perineum. I do not hesitate to do an episiotomy for a premature infant in a primipara. It is much better to have a 2-inch incision in the perineum than a tear in the falx cerebri.

Keeping the perineum intact has become an obsession with many midwives and doctors. The teaching of the evils of perineal tear has been overdone. Every midwife feels that the biggest disgrace in a delivery is a perineal tear, with the result that sometimes the foetal head is subjected to so much pressure in an attempt to maintain flexion and a slow delivery that a 'cerebral' baby is born. But the midwife or doctor has the boast of an intact perineum. What does a small tear in the perineum really matter? What does matter is a healthy baby. A small tear can be stitched within minutes.

Credé's Expression

Credé's method is still used on conscious patients, in spite of the fact that this way of expressing the placenta is universally condemned. It is indeed a distressing experience watching an outsize doctor or midwife grasping the uterus and trying to squeeze the placenta out in spite of the woman's groans of protest. One has only to see the resulting profound shock, or a woman bleeding from an inverted uterus as a result of this manoeuvre, to appreciate its danger. Credé's expression should only be attempted under general anaesthesia, and then only once.

Toxaemia of Pregnancy

There are 2 points worth mentioning. It is most important to check the blood pressure when a patient is first seen. Very often this essential feature is left until the patient is 7 months pregnant or more. Many fail to appreciate that a hypertensive toxaemia, i.e., a toxaemia superimposed on an essential hypertension, carries a far worse prognosis for the infant than a pure toxaemia (pre-eclamptic toxaemia).

The other point is this: A patient manifesting features of a mild toxaemia is often sent home and told not to eat salt. The patient, and often the doctor does not appreciate the significance of the words. In these cases, what is intended is that the intake of sodium should be restricted. It is most important then that the patient should not take any antacid preparation containing sodium. She is told not to eat salt, and thinks in terms of the salt cellar—but the doctor should point out that foods like ham, bacon, corned beef, smoked fish, are salt foods rich in sodium and therefore to be avoided.

Caesarean Section

Those of us who are practising in the smaller towns must accept the fact that classical Caesarean section has given way to the lower-segment operation. It must be most disconcerting to the young houseman who at his medical school was taught all the advantages of the lower-segment Caesarean section to start work at a hospital and see only the classical approach. But he is impressed by the apparent ease and drama of the classical section, and when he starts out on his own, the classical section may be perpetrated. Therefore I cannot but feel that it is well worth while repeating the advantages of lower-segment Caesarean section and the reasons why it has established itself as the operation of choice:

1. The incision is well away from the placental site and the uterine sinuses, so that less bleeding occurs.
2. It is the part of the uterus which involutes slowly; hence a stronger scar will form and be less likely to rupture.
3. The scar is low down and covered by peritoneum and, therefore, there is less chance that adhesions will form.
4. It is far easier to suture because of the thinness of the uterus in this area.
5. The incision is directly over the foetal head, so that the head is far more easily delivered by a scoop of the hand, and it is easier to apply Wrigley's forceps should it be necessary to deliver the head in this way.

I do not completely condemn the classical operation, and I still feel it is justified in conditions such as the following:

(*a*) Neglected shoulder presentation where the liquor has drained away and the uterus is in tonic contraction on the foetus.

(*b*) Where there is likely to be difficulty in gaining access to the lower segment because of adhesions of fibroids.

(*c*) The presence of placenta praevia.

While on the subject of Caesarean section I should like to mention where considerable time can be saved in the operation. It should be the primary concern of the surgeon and anaesthetist to produce a crying infant. The purpose is to avoid apnoea in the infant, and, as a general practitioner who is also called upon to give many anaesthetics, I find nothing more exasperating than to watch a surgeon waste 10-15 minutes in tying off superficial skin bleeders.

Manual Removal of Placenta

This a far too often done as a routine procedure after the application of forceps. Having seen the dangers of this method—sepsis or rupture of the uterus—I feel that it should only be done as a last resort.

An alternative and, in my opinion, a more satisfactory procedure is an intravenous injection of 0·5 mg. of ergometrine given when the head is born. When delivery is completed the cord is grasped in the right hand and a firm steady pull is applied while the left

hand carries out a rubbing-up movement of the uterus. Again I have seen a complete placenta delivered in this way.

Blood Transfusion

In no branch of medicine does blood loss occur so dramatically and in such large volume as in obstetrics, and after the haemorrhage the woman will be more subject to infection and her recovery back to normal will be slow. However, by transfusion with whole blood the general health of these women is quickly restored.

In the smaller towns we are less fortunate than our colleagues in the cities. We have not got skilled technicians at our service 24 hours a day, and we therefore have to be doubly careful when administering blood. It is necessary to remember the following points:

1. The correct technique must be used and great care taken in cross-matching donors' cells against recipients' serum; otherwise fatalities may occur.
2. Infected blood, owing to haemolysis, looks like red ink, and the supernatant fluid is reddish instead of pale yellow. Such blood should of course not be administered. Dangerous infection however, may be present in the blood without these gross signs.
3. Blood which has been cooled down excessively may be haemolysed, and is dangerous to use.
4. Blood which has been heated above 40°C may also be haemolysed, and dangerous to use.
5. The giving of too much blood, and too quickly, is dangerous especially in cases of anaemia of long standing, and in old people. The heart muscle cannot deal with the excessive amount of blood and acute pulmonary oedema may result.
6. No Rh negative woman should ever get Rh positive blood. The South African Blood Transfusion Service will group and test the Rh factor free.

A baby developing jaundice after the first 24 hours is unlikely to be a case of haemolytic disease of the newborn.

Puerperal Pyrexia

I should like to give a scheme of approach in the examination of patients with temperatures during the puerperium:

1. *Uterine and Vaginal Sepsis.* All lacerations should be sutured and blood clots cleaned away after delivery or any obstetrical manoeuvre. The anaerobic streptococcus will multiply rapidly in the presence of blood clots. A sudden rigor with temperature and headache early in the puerperium is very suggestive of haemolytic streptococcal infection. Both these organisms respond well to penicillin. The character of the lochia and the rate of uterine involution, will give a good indication of the source of infection.
2. *Respiratory-Tract Infections.* Examine (*a*) the throat and (*b*) the chest, for pneumonia or small areas of collapse, particularly if an aneasthetic has been administered.
3. *Thrombosis.* Examine the legs for thrombophlebitis or phlebothrombosis. A woman developing signs of white leg will respond dramatically to paravertebral block.
4. *Renal Tract.* If a temperature develops from the 6th day onwards, it is well worth while to examine the urine for pus.
5. *Engorgement of Breasts.* This rarely produces a temperature of more than 100°F.
6. *Megaloblastic Anaemia.* This should be remembered as a cause of pyrexia, especially when dealing with the poorer sections of the community.

OFFICIAL ANNOUNCEMENT : AMPTELIKE AANKONDIGING

MEDICAL AID SOCIETIES

The following new Medical Aid Societies were approved by Federal Council at its meeting held at Vereeniging on 11-13 April 1956:

1. Anglo-Alpha (Dudfield) Medical Aid Society, P.O. Box 7727, Johannesburg.
2. Federated Employers' Medical Aid Society, P.O. Box 666, Johannesburg.
3. Federation of Master Printers of S.A. Medical Aid Society, P.O. Box 1200, Johannesburg.
4. The General Motors Medical Aid Scheme, P.O. Box 1137, Port Elizabeth.
5. Joseph Liddle (Pty.) Ltd. Medical Aid Society, P.O. Box 106, Johannesburg.
6. Sun Insurance Office Ltd. Staff Medical Aid Fund, P.O. Box 429, Johannesburg.

A complete list of approved societies will be published in the *Journal* shortly.

L. M. Marchand
Associate Secretary

Medical House
Cape Town
18 April 1956

MEDIESE HULPVERENIGINGS

Op sy vergadering van 11-13 April te Vereeniging gehou het die Federale Raad onderstaande nuwe Mediese Hulpverenigings goedgekeur:

1. Anglo-Alpha (Dudfield) Medical Aid Society, Posbus 7727, Johannesburg.
2. Federated Employers' Medical Aid Society, Posbus 666, Johannesburg.
3. Federation of Master Printers of S.A. Medical Aid Society, Posbus 1200, Johannesburg.
4. The General Motors Medical Aid Scheme, Posbus 1137, Port Elizabeth.
5. Joseph Liddle (Pty.) Ltd. Medical Aid Society, Posbus 106, Johannesburg.
6. Sun Insurance Office Ltd. Staff Medical Aid Fund, Posbus 429, Johannesburg.

Binnekort sal 'n volledige lys van goedgekeurde verenigings in die *Tydskrif* verskyn.

L. M. Marchand
Medesekretaris

Mediese Huis
Kaapstad
18 April 1956

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

Glaxo Laboratories announce *Suavitil* (benactyzine hydrochloride) which they state provides a new approach to the treatment of psychoneuroses. It has an effect upon the central nervous system differing from that of substances previously used in the treatment of these conditions. It can help to resolve a neurosis by a selective action which involves no classical sedative or hypnotic effect of the barbiturate type, nor any general suppressive effect such as that produced by some of the more recently introduced compounds. The patient remains alert and objective during treatment.

Suavitil is a member of the group of anticholinergic substances, several of which have selective activity upon various functions of the brain. There is a wide margin of safety between therapeutic and toxic doses. It has no detectable chronic toxicity in animals receiving large doses daily for periods exceeding a year. Patients on high dosage for several months have shown no signs of organic damage.

When given in therapeutic dosage the substance is eliminated from the body within a few hours, so there is no practical likelihood of cumulative effect. It produces no euphoria and no withdrawal symptoms have been detected.

It raises the threshold for external stimuli, so that the patient in a state of tension arising from excessive strain no longer reacts

to stimuli which previously would have provoked marked irritation. In therapeutic doses the substance eliminates mental rumination and chaotic speculation, which may be acting as an obstacle to organized thinking.

Suavitil has valuable application in psychoneuroses arising from external stress. It seems to be particularly effective in psychoneuroses with anxiety, but can be effective also in psychoneuroses with depressive and obsessive-compulsive reaction.

Tentative experience suggests that the substance may be useful in certain psychosomatic disorders. It seems to have useful potential application in the treatment of compulsive alcoholism.

Suavitil does not replace the need for psychotherapy where that is indicated. Rather, it should be employed to supplement psychotherapy and often appears to render the patient more amenable to this form of treatment.

A dose of 1 mg. or 2 mg. is usually effective, although in some instances 3-mg. doses may be needed. In special circumstances doses of 10 mg. have been given. Because the substance is rapidly eliminated, the dose must be repeated three or four times daily.

* * *

Maybaker (S.A.) (Pty.) Ltd. announce the introduction of '*Transithal*' (buthalitone sodium), an intravenous short-acting anaesthetic. The smooth induction and extremely short recovery period afforded by this product make it of particular value for use in out-patients and casualty departments and for dental and other procedures requiring brief intravenous anaesthesia with minimal post-anaesthetic care of the patient.

'Transithal' is available in cartons of 5 × 1 gramme ampoules together with 5 × 10 c.c. ampoules of Water for Injection.

PASSING EVENTS : IN DIE VERBYGAAN

Dr. John Godfrey Cowley, M.B., B.Ch., D.T.M. and Hy. (Rand) has been elected a Member of the Royal College of Physicians of Edinburgh.

* * *

Dr. Paul Marchand, Ch.M., F.R.C.S., of the Department of Surgery of the University of the Witwatersrand has been awarded the Jacksonian Prize by the Council of the Royal College of Surgeons of England for a treatise on the subject of diaphragmatic hernia.

* * *

Association News. An ordinary general meeting of the Queenstown Division (Border Branch) of the Medical Association of South Africa was held at the Frontier Hospital, Queenstown, on 23 February at 8 p.m. Dr. M. I. Papilsky presided, and 11 other members were present. Discussions took place on Medical Aid Societies, Benefit Societies, and on blood transfusion. Dr. A. Rosin presented a paper on 'Common Obstetric Problems' (see page 407 of this issue of the *Journal*).

* * *

Quiz Evening on Alcoholism. The South African Society of Industrial Health and the Southern Transvaal Subgroup of the Association of Physicians are holding a combined meeting on Thursday, 3 May 1956 at 8.15 p.m. at Medical House, Esselen Street, Johannesburg. The meeting will be in the form of a quiz evening. The subject is Alcoholism.

The Forum will consist of the following:

Prof. G. A. Elliott, Medicine. Dr. Max Feldman, Psychiatry. Dr. B. J. P. Becker, Pathology. Mr. C. A. L. Warffemius, Psychology. Dr. S. Biesheuvel, Personal Management and Human Relations. Dr. D. F. van der Merwe, Social Welfare. Dr. R. A. Mathews, Medical Practice. Dr. H. A. Shapiro. Medico-legal.

The address to which questions should be sent is: Hon. Joint Secretaries, Industrial and Physicians Groups, Medical House, Esselen Street, Johannesburg.

* * *

Research Forum, University of Cape Town. There will be no Research Forum nor Clinico-Pathological Conference on Tuesday, 1 May. There will be a meeting of the Research Forum in the A-floor Lecture Theatre, Groote Schuur Hospital, on Tuesday, 8 May 1956, at 12 noon. *Speaker:* Dr. R. Hoffenberg. *Subject:* Gonodal Dysgenesis in Relation to Current Theories on Hermaphroditism.

* * *

Dr. James Marshall, M.D., Dermatologist, formerly of Johannesburg and Pretoria, is now in practice at 610 Netherlands Bank Buildings, St. George's Street, Cape Town. Telephones, Rooms 3-1416, Residence 8-4054.

* * *

The annual dinner-dance of the Northern Transvaal Branch of the Medical Association of South Africa is being held at the Pretoria Country Club at 8 p.m. on 12 May 1956. Tickets will be £2 2s. 0d. per couple, and a buffet supper will be served from 8 to 9.30 p.m.

BOOKS RECEIVED : BOEKE ONTVANG

Medical Terms—Their Origin and Construction: By Ffrangcon Roberts, M.A., M.D., F.F.R. Pp. viii + 88. 6s. net. London: William Heinemann Medical Books, Ltd. 1956.

Sick Children—Diagnosis and Treatment: 7th Edition. By Donald Paterson. 7th Edition revised by Reginald Lightwood and F. S. W. Brimblecombe. Pp. x + 593. 42s. London: Cassell and Company Ltd. 1956.

Hydrocortisone in Orthopaedic Medicine: By James Cyriax, M.D., M.R.C.P. Pp. 31. 5s. London: Cassell and Company Ltd. 1956.

Midwifery: Principles and Practice for Pupil Midwives, Teacher Midwives and Obstetric Dressers. Fourth Edition. By R. Christie Brown, Barton Gilbert, Donald B. Fraser and Richard H. Dobbs. Pp. viii + 892. 25s. net. London: Edward Arnold (Publishers) Ltd. 1956.

Diseases of the Nose, Throat and Ear: A Handbook for Students and Practitioners. Sixth Edition. By I. Simson Hall, M.B., Ch.B., F.R.C.P.E., F.R.C.S.E. Pp. xii + 463, with 8 coloured plates. 20s. plus net 1s. postage abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1956.

Yellow Fever Vaccination: By Smithburn *et al.* Geneva 1956. (World Health Organization: Monograph Series No. 30), 238 pages, 37 figures, select bibliography, index. £1 5s., 85·00, or Sw. fr. 15. French edition in preparation.

A Practical Handbook of Psychiatry for Students and Nurses: Third Edition. By Louis Minski, M.D., F.R.C.P., D.P.M. Pp. 144. 7s. 6d. net. London: William Heinemann—Medical Books—Ltd. 1956.

Christopher's Textbook of Surgery: Sixth Edition. Edited by Loyal Davis, M.D. Pp. xvii + 1484, with 1,359 illustrations on 716 figures. $15·50. Philadelphia and London: W. B. Saunders Company. 1956.

Poliomyelitis: Second Edition. By W. Ritchie Russell. Pp. xi + 147. 16s. net. London: Edward Arnold (Publishers) Ltd. 1956.

1955–1956 *Year Book of Drug Therapy.* Edited by Harry Beckman, M.D. Pp. 560. $6·00 post paid. Chicago: The Year Book Publishers Inc. 1956.

Current Therapy—1956. Latest Approved Methods of Treatment for the Practising Physician. Edited by Howard F. Conn, M.D. Pp. xxx + 632. $11·00. Philadelphia and London: W. B. Saunders Company. 1956.

Doctor and Patient and the Law. Third Edition. By Louis J. Regan, M.D., LL.B. Pp. 716. South African Price £5 6s. 3d. St. Louis: The C. V. Mosby Company. 1956.

A Manual of Anaesthetic Techniques. By William J. Pryor, M.B., Ch.B. (N.Z.), F.F.A.R.C.S. (Eng.), D.A. (Eng.). Pp. viii + 224, with 77 illustrations. 27s. 6d. Bristol: John Wright & Sons Ltd. 1956.

Pulmonary Circulation and Respiratory Function. A Symposium held at Queen's College, Dundee. Pp. 44. 12s. 6d. net, plus 10d. postage abroad. E. & S. Livingstone Ltd. Edinburgh and London. 1956.

Practical Urology: Case Comments and Late Results. By Alex E. Roche, M.A., M.D., M.Ch. (Camb.), F.R.C.S. (Eng.). Pp. xii + 258, with 132 illustrations. £1 15s. net. London: H. K. Lewis & Co. Ltd. 1956.

Rassenkruising bij de mens. By Dr. J. W. Bruins. Pp. 51. Fl·40. Arnhem: Drukkerij D. Derksen. 1955.

The Treatment of Eczema in Infants and Children. By Lewis Webb Hill, M.D. Pp. 79, with 39 illustrations. £1 14s. 0d. St. Louis: The C. V. Mosby Company. 1956.

CORRESPONDENCE : BRIEWERUBRIEK

'MEDICAL JOURNALS IN SOUTH AFRICA'

To the Editor: With reference to the Editorial[1] under this title in your *Journal* of 7 April 1956, it is evident that medical publications in South Africa fought a grim struggle for survival, and after many years, this journal has evolved as the official organ of the medical profession in South Africa.

With this in mind, I am puzzled to understand why only fleeting reference was made to all other current South African medical publications. Surely these publications must be complimented on their boldness, their pioneering work and for the purpose they serve.

Whereas past editors of defunct medical journals are mentioned and their work described, no mention is made of any former living South African editor. Why was mention deliberately omitted of Dr. H. Shapiro, a former editor of this very journal whose heritage was printed in the Editorial.

Surely this journal, the *Paterfamilias* of all medical publications in South Africa could show its greatness by more generous mention of such journals as *Medical Proceedings*, *The South African Journal of Medical Sciences* and others. What is more, students' publications like *The Leech* have been completely omitted.

Could it be that a fear exists that because the *South African Medical Journal* is 'thrust upon us', it needs an appraisal in the light of competitive private publications.

It would be interesting to know what success this journal would claim if it depended on voluntary subscription.

A very useful purpose would have been served if the Editorial Board had listed every current South African medical publication, a word about its editor and the purpose of the publication. And, what is more, such information is most relevant under the heading *Medical Journals in South Africa.*

J. Fine

204 Medical Centre
Jeppe Street
Johannesburg
19 April 1956

[It should be noted that the Editorial in question was one of two articles[1,2] that were historical in nature, and that, as our correspondent observes, no editor still living was mentioned.—*Editor.*]

1. Editorial (1956): S. Afr. Med. J., **30**, 337.
2. *Idem* (1956): *Ibid.*, **30**, 311.

'ONTHAAL-ONKOSTE', NAGRAADSE STUDIE, EN INKOMSTEBELASTING

Aan die Redakteur: Dit het onder my aandag gekom dat geneeshere toegelaat is om sogenaamde 'onthaal-onkoste' tot 'n bedrag van ongeveer £150 per jaar af te trek van die inkomste wat vir belasting aangeslaan word. Met enkele uitsonderings, bv. waar 'n geneeshere noodgedwonge gaste van die buiteland of ander dele van die land, wat lesings kom hou moet onthaal, kan ek nie insien hoe 'n geneeshere van hierdie aftrekking gebruik kan maak nie. Ek neem aan dat die Ontvanger van Inkomste veronderstel dat die 'onthaal-onkoste' aangegaan is met die bedoeling om die praktyk van die betrokke persoon uit te brei of in stand te hou. Indien dit wel die geval is, lyk dit vir my of geen geneesheer (met die enkele uitsonderings hierbo genoem) van die aftrekking gebruik sal kan maak nie, omdat dit oneties sou wees om deur middel van traktering van óf pasiënte óf ander praktisyns 'n mens se praktyk te probeer verbeter. Ek sal dit op prys stel as enige van u lesers my kan aandui onder watter omstandighede geneeshere geregtig sou wees om op hierdie 'onthaal-onkoste' aanspraak te maak, sonder om oneties te handel.

Ek verneem ook dat daar deur die Mediese Vereniging vertoë tot die Minister gerig gaan word in verband met onkoste wat aangegaan word in verband met nagraadse studie. Myns insiens kan die feit dat bogenoemde 'onthaal-onkoste' toegestaan word, deur die deputasie wat die Minister sal ontmoet aangehaal word in verband met hierdie saak. My redenering is dat as die Ontvanger van Inkomste bereid is om onkoste wat aangegaan word in verband met traktering toe te laat, hy des te meer onkoste wat in verband met nagraadse studie aangegaan word, moet toelaat, omdat laasgenoemde die individuele praktisyn en die beroep as geheel (en daarom ook die land) tot voordeel kan strek en aangemoedig behoort te word.

Ek sal selfs so ver gaan as om voor te stel dat daar gevra moet word dat onkoste in verband met nagraadse studie toegelaat word in die plek van s.g. 'onthaal-onkoste', maar ek begryp dat dit miskien 'n punt is waaroor daar onder geneeskundiges nie eenstemmigheid sal wees nie. In verband met onkoste vir nagraadse studie is daar nog 'n belangrike punt, nl. dat onkoste in verband met buitelandse nagraadse studie besonder hoog is. Daarom sal dit wenslik wees om te versoek dat die bedrag wat aftrekbaar sal wees, oor 'n paar jaar kan ophoop indien dit vir buitelandse studie gebruik word.

J. K. Bremer

Van Riebeek Mediese Gebou 409
Schoemanstraat 295
Pretoria
5 April 1956

CORONARY HEART DISEASE AND DIETARY FAT

To the Editor: Your recent editorial[1] on this subject (issue of 17 March) was read with great interest by those of us actively engaged in research work bearing on degenerative heart disease. We would like to comment on some of the points you mentioned.

1. *Age*

Although coronary heart disease is mainly confined to middle age and old age, we think that it is insufficiently appreciated that the disease is the major health threat to all males *over* 30 *years.*

2. *Sex*

That the disease is commoner in men than in women, at least until late middle age, is well known. We are glad that you have pointed out that, notwithstanding the foregoing, coronary thrombosis is commoner in females of privileged communities than in men in under-privileged communities.

3. *Cellulose Intake*

We were interested to note your reference to differences in cellulose intake as being one of the differences between modern sophisticated diets and those consumed by poorer communities. Some time ago, one of us suggested that the level of intake of this factor, directly or indirectly, may well have some aetiological importance in atherosclerotic disease. It is not that we attach undue importance to cellulose *per se*, but rather to that pattern of diet (and concomitant metabolic pattern and ramifications, e.g. in bowel motility) in which a high content of cellulose or crude fibre is a predominant feature. Now, the diet not only of the Bantu, but of Western populations a few generations ago, and of certain White populations during war-time, included a high cellu-

lose intake; and among consumers, a low or reduced incidence of atherosclerosis is believed to have obtained. But why single out and consider that level of *cellulose* intake may be of relevance? Diets high and low in cellulose content give rise to different intestinal flora. A diet very high in coarse cereal products, i.e. high in cellulose, is accompanied by the voiding of large amounts of faecal fat, up to 22 grams per diem; such fat is deemed to be mainly of endogenous origin. Furthermore, the excretion of sterolic compounds in the stools is known to be much greater during the consumption of high compared to low cellulose diets. There can be no doubt, therefore, that there are differences in fat metabolism in diets of different cellulose intake, so that differences in cholesterol metabolism are only to be expected. Absolutely apart, therefore, from the type and amount of fat consumed, level of intake of cellulose may well have considerable influence on the over-all lipid metabolism picture. Unfortunately, in relation to diets of different cellulose content, it has to be admitted that precise knowledge of such aspects as cholesterol excretion into the bile, absorption and re-absorption, destruction by intestinal flora, and loss in the stools, is virtually nil. Incidently, it is interesting to note that diets high in cellulose have been correlated not only with a low incidence of atherosclerosis, but also with a low incidence of appendicitis, eclampsia, cholelithiasis, and peptic ulcer.

4. *Animal and Vegetable Fat*

For some time many of us have had reservations regarding the view of Ancel Keys that animal and vegetable fats have approximately equal effect in regulating serum cholesterol concentration. The results of several experimental studies on humans have provided no support for this view, neither have field observations undertaken on groups of persons habituated to diets high in animal or vegetable fats. There is now ample evidence which demonstrates that animal vegetable fats differ markedly in cholesterolaemic capacity, and more recently Kinsell has suggested that the explanation of the difference may lie, not so much in whether fats are of animal or vegetable origin, but rather in the degree by which they are saturated or unsaturated, i.e. the proportion of double bonds present in the carbon chains of the fatty acids or esters. The Cape Town workers under Professor Brock are to be highly congratulated on their excellent demonstration of the importance of the degree of saturation of the fats in cholesterolaemic capacity. The findings of these workers open an enormous field for the investigation of short-term and long-term effects of various unsaturated concentrates, pure fats and fat-containing foods, on the blood-fat components of both Bantu and European subjects. Certain of the above studies are about to be initiated in collaboration with the Department of Medicine of Pretoria University, the National Nutrition Research Institute, and ourselves. On the laboratory side, we are now engaged in determining iodine values (which provide a direct measure of degree of saturation) on total fat, fatty acids, and higher fatty acids in the serum of Bantu and European subjects of different age-groups. In addition, we are making comparisons of the iodine number of fat extracted from aortic tissue from Bantu and European subjects of different age-groups.

5. *Serum-Cholesterol Levels*

There is now no doubt that *population groups* characterized by low fat intakes, are likewise characterized by low serum cholesterol concentrations, and a low incidence of atherosclerosis. Conversely, population groups marked by high fat intakes frequently, although not invariably, have high serum concentrations, and are much more prone to develop atherosclerosis. But when one comes to individuals, the correlation is far less satisfactory. Recently we have encountered relatively low cholesterol-levels in several patients who have had attacks of coronary thrombosis. In such subjects the important point may well be not so much the absolute level of serum cholesterol obtaining, but rather the steepness of the rise which occurred during the months or years before the attack. Indeed, Professor Frederick Stare, of Harvard University Medical School, who recently visited us, and who is an authority on both experimental and human atherosclerosis, averred that there was something to be said for all adults to have annual determinations of their serum-cholesterol level. Now, in our experience, the serum-lipoprotein picture taken together with the serum-cholesterol concentration is more informative than the latter alone. We have almost invariably noted a high beta-lipoprotein concentration in patients with coronary heart disease. But even in relation to this aspect there are anomalies; e.g. the Gautemalans are characterized by a relatively low serum-cholesterol concentration (almost identical to that found in Bantu adults), yet the lipoprotein picture is closely similar to that of Northern Americans. The incidence of severe atherosclerosis in Guatemalans is believed to be low.

Reference may be made to a further criterion. The school of which Goffman is the chief protagonist some years ago put forward their conception of an *atherogenic index*, calculated from data on the flotation rate of the lipoproteins; it is maintained that this index is of value in seeking to differentiate between coronary and non-coronary subjects. Many workers, however, do not agree that the index is of more diagnostic value than knowledge of cholesterol level plus lipoprotein picture. In an endeavour to clarify the situation, at least in as far as the Bantu is concerned, we are carrying our determinations of serum cholesterol, the electrophoretic lipoprotein picture, and flotation rate of the lipoproteins (in collaboration with Dr. F. Joubert of the C.S.I.R. National Chemical Research Laboratory at Pretoria), in serum from Bantu and European subjects of different age-groups and, in the Europeans, in subjects with and without clinical evidence of coronary thrombosis.

Much of the present-day work relates primarily to bloodlipid components, and these, we all hope, and not without reason, are closely involved in regulating the development of atherosclerosis. By dietary manipulation we can, without much difficulty, alter the bloodlipid picture. But whether the changes are accompanied by a retardation of lesions in the blood vessels will become apparent only after years of study, and by the careful compilation of vital statistics, etc. One of us has suggested that while it is right to be hopeful, caution should be exercised and it should be borne in mind that dietary pattern may well be of importance. In other words while one can, by alterations in diet, acquire a bloodlipid picture resembling that of the Bantu, the diet consumed is still markedly different from that of these people. This gives rise to the question, must a favourable bloodlipid picture lie within a particular dietary framework before leading to a retardation of atherosclerosis?

The laboratory study of coronary heart-disease includes, of course, numerous aspects in addition to those mentioned. The precise pathology of the vascular system seen at necropsy (including the total chemical composition of whole aorta) requires to be correlated with the total-lipid picture determined on post-mortem blood. There are studies on heparin and lipoprotein lipase, the role of pancreatic elastase, the endocrine pattern, and also blood-coagulation factors, in Bantu and European subjects, and so forth. On certain of these aspects, research work is proceeding in this centre.

Recently, the Ernest Oppenheimer Research Unit in Cardiovascular Disease, made it possible for one of our assistants, Miss M. Andersson, to visit several universities and research institutes in the United States and in Britain where research work in atherosclerosis is in progress. One of her main impressions was that overseas workers believe that the most important contribution that South Africa can produce is to define as closely as possible the relevant biochemical, pathological and other differences between the Bantu and White populations—investigations which will occupy workers, whether in Cape Town, Pretoria or Johannesburg, for some considerable time to come. We are hopeful that the elucidation of the age-trend of various changes in both populations may afford information of aetiological importance, or at least of value in the earlier diagnosis and the control of the disease.

This letter is published with the permission of the Council for Scientific and Industrial Research.

I. Bersohn

Ernest Oppenheimer Unit for Research in Cardio-vascular Diseases, South African Institute for Medical Research

A. R. P. Walker

Human Biochemistry Research Unit of the Council for Scientific and Industrial Research and the South African Institute for Medical Research

J. Higginson

Geographical Pathology Unit of Baragwanath Hospital and the South African Institute for Medical Research.

Johannesburg

31 March 1956.

1. Editorial (1956): S. Afr. Med. J., **30**, 259.